Modern Concepts of Cardiovascular Disease

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FACTORS ENTERING INTO THE PROGNOSTIC EVALUATION OF THE PATIENT WITH ESSENTIAL HYPERTENSION

Patients with essential hypertension have in common an elevated diastolic pressure of unknown etiology. Yet they may pursue widely divergent clinical courses at different rates of progress. Some patients may have hypertension for several decades and show minimal organic damage, while others may succumb after a few years to heart failure, coronary disease, or cerebral hemorrhage. The importance of an evaluation of this direction-velocity vector is self-evident. Unfortunately, at the present stage of our knowledge, prognosis and actual eventuation are not yet congruent.

In terms of gross statistical probabilities, hypertension shortens life and produces death most frequently from heart disease; a smaller percentage of hypertensives die of cerebral hemorrhage, and a minority succumb to uremia. In addition, however, there are certain factors, consideration of which sometimes enables us to modify our concept of the future course of the "average" hypertensive to conform more closely to that of the individual patient.

Sex, Race, and Age of Onset

Although the incidence of essential hypertension is higher in women, they are better able than men to tolerate the disease. This may be due, in part, to the lower incidence of coronary disease in females. Other factors are doubtlessly at work as indicated by the fact that far fewer women than men enter the malignant phase. It is also recognized that essential hypertension is more apt to run a severe course in the colored races.

Generally speaking, when essential hypertension appears early in life, it tends to be more severe and rapidly progressive. Younger patients comprise the majority of the small group of hypertensives whose disease undergoes malignant transformation. Individuals who do not develop hypertensive pressures until after they have passed the half-century mark frequently exhibit a mild form of the disease.

Family History

Mere occurrence of hypertensive disease in a patient's blood relatives may be of little prognostic value. Severity of the disease, as well as familial incidence, must be taken into account. When complications and/or death occur at an early age in several of a patient's blood relatives, such a family history has unfavorable portent.

Duration of the Disease

An individual who has been hypertensive for many years and who shows little organic damage is apt to continue this benign slowly-progressive course. Similarly, a rapidly advancing disease does not usually diminish its velocity. However, such extrapolations based on previous rate of progress are not always reliable and exceptions are notable.

Height of the Blood Pressure

It is difficult to establish a correlation between the height of the blood pressure, as determined by office visits, and the severity of the disease. Patients with extremely high diastolic pressures have been known to pursue a benign course for many years. However, blood pressure is exceedingly variable, and office blood pressures are therefore an unreliable index of the actual height of the pressure throughout the 24-hour period. It is common observation that patients with *sustained* diastolic pressures of over 130 mm. Hg. do not long maintain their cardiac compensation. In the sense of total 24-hour pressure, over a period of time, it can be said that patients with higher diastolic levels do not fare as well as those whose diastolic pressures are lower.

Similarly, hypertensive encephalopathy and the malignant syndrome commonly follow periods of excessive diastolic pressure. A progressive increase in the diastolic level is an unfavorable sign.

It is doubtful if the systolic pressure *per se* is of importance in determining the rate of progress of the vascular lesions.

Lability of the Blood Pressure

A patient whose blood pressure undergoes wide fluctuations usually has a more benign form of the disease than one whose pressure is high and relatively inflexible. This phenomenon may be explained by difference in extent of vascular sclerosis. The individual whose pressure is maintained by increased peripheral resistance due largely to arteriolar vasospasm is theoretically in an earlier and more reversible stage of the disease than one whose increased peripheral resistance is due chiefly to irreversible organic arteriolosclerosis..

This principle probably applies not only to spontaneous blood pressure fluctuation, but also to the hypotensive effects of various procedures such as the amytal test, tetraethylammonium ion injection, and bed rest.

Nutritional Status

It is well known that obese hypertensives show considerable improvement in their cardiac status and height of blood pressure after losing excess weight. In this sense, the outlook for the obese patient is better than for the patient in a comparable stage of the disease who does not possess excess adiposity, simply because the former possesses the potentiality for removal of a major health hazard.

Organic Change

Of greatest importance in evaluation of direction of hypertensive disease is assessment of the organ damage that has occurred. Velocity may be estimated by referring the amount of this change to the time interval during which it was produced.

(1) *Arterioles*

Although hypertension probably accelerates atherosclerosis, the predominant vascular damage produced by elevated arterial diastolic pressure is sclerosis of the arterioles. The arterioles of the ocular fundi are the only ones in the body that may be examined directly without recourse to biopsy. Although exceptions occur, the fundal arteriolar changes, in general, parallel the changes in the remainder of the arteriolar bed. It is this fact plus the empirical correlation between retinal lesions and life expectancy, that make ophthalmoscopic examination of prime value in hypertensive disease.

It is convenient to divide the fundal arteriolar changes into two types: reversible and irreversible. The former appear first and consist of attenuation, narrowing, and spasm. The latter consist of variability of, and increase in the arteriolar light reflex, venous compression at the arteriovenous crossings, and arteriolar tortuosity and irregularity. These are permanent, irreversible changes due to sclerosis. When the sclerotic changes are pronounced, the hypertension proves to be almost always of long duration.

(2) *Retinae*

Retinal hemorrhages and fluffy exudates, when due to hypertension, bespeak a much severer disease and a shorter life expectancy. However, these lesions are completely reversible and may disappear entirely when blood pressure is lowered.

Edema of the optic disc is an ominous sign. In hypertensive disease, it usually denotes increased intracranial pressure and occurs in hypertensive encephalopathy and in the malignant phase of essential hypertension.

Patients with the full-blown picture of hypertensive neuro-retinopathy rarely live more than one to two years after its appearance.

(3) *Cerebral area*

Persistent and severe headaches may presage hypertensive encephalopathy and as such are of ill portent. History of previous stroke usually indicates not only an advanced stage of the disease, but also that the cerebral area is a vulnerable one.

Although cerebral hemorrhage accounts for only 15% of deaths in essential hypertension, it is regarded by the laity as one of the chief menaces of the disease. This Sword of Damocles by no means threatens all hypertensive patients alike—some are more prone to suffer cerebral hemorrhage than others. The combination of many retinal hemorrhages (in the absence of papilledema and exudates), frequent epistaxes, severe occipital headaches, motor or sensory disturbances, and episodes of vertigo or syncope indicate that a patient is more likely to suffer a stroke than one in whom these findings are absent.

(4) *Cardiac area*

Impairment of cardiac function constitutes one of the major depredations of hypertensive disease. Establishment of the cardiac area as a *locus minoris resistentiae* operates either via failure of compensation or failure of the coronary circulation. The former may be detected by the signs and symptoms of diminution of cardiac reserve, cardiac dilation (as shown by physical examination and teleoroentgenogram), cardiac hypertrophy (as indicated chiefly by electrocardiogram), and overt evidence of heart failure. Inadequacy of the coronary circulation reveals its presence by producing angina pectoris and electrocardiographic evidence of coronary insufficiency. The response of the heart by strain to the stresses of hypertension indicates early failure of adaptation of the organism to the disease. Generally speaking, the more extensive the strains, the poorer the prognosis.

(5) *Renal area*

It is a rare patient with essential hypertension whose kidneys become sufficiently decompensated to constitute a cause of death. When this occurs, it does so almost exclusively in the malignant phase. Extensive renal involvement as evidenced by severe depression of renal function should arouse suspicion that the disease has undergone malignant transformation. As such the prognosis is almost hopeless. It should be remembered, however, that albuminuria and marked diminution of kidney function may occur in heart failure alone.

Socio-economic Factors

In hypertensives as in normotensives, excessive activity, nervous tension, and emotional unrest tend to raise blood pressure. Theoretically at least, these factors can operate to increase the vascular stress. Furthermore, as in cardiac disease of other etiology, when economic exigencies prevent the hypertensive cardiac from obtaining the optimum amount of rest, the heart disease is intensified and the outlook becomes accordingly poorer.

It is readily apparent, therefore, that the prognosis in essential hypertension is determined by many factors, not all of which are known. It may be difficult to assay all these factors at a single office visit, or even during a short period of hospitalization. Often, many months of careful observation are required before a prognostic evaluation can be made. Even then, the limitations of our present knowledge and methods are all too frequently brought sharply into focus by the vagaries of the natural history of the disease.

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